	Hits	Search Text	DBs	Time Stamp
1	1713	sendai adj vir$2 or hemagglutinating adj virus	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/01/10 16:10
2	1600	sendai adj vir$2 or hemaglutinating adj virus	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/01/10 16:10
3	13	(sendai adj vir$2 or hemagglutinating adj virus) with gene adj therapy	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/01/10 16:11
4	0	(("20020081706") or ("20020100066")).PN.	USPAT	2003/01/10 16:18
5	0	"20020081706" or "20020100066"	USPAT	2003/01/10 16:19
6	410	asakawa.in.	USPAT	2003/01/10 16:19
7	2	"20020081706" or "20020100066"	USPAT; US-PGPUB	2003/01/10 16:19

	Hits	Search Text	DBs	Time Stamp
1	1713	sendai adj vir$2 or hemagglutinating adj virus	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/01/10 16:10
2	1600	sendai adj vir$2 or hemaglutinating adj virus	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/01/10 16:10
3	13	l1 with gene adj therapy	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/01/10 16:11

(FILE 'HOME' ENTERED AT 14:27:27 ON 10 JAN 2003)

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE' ENTERED AT 14:27:45 ON 10 JAN 2003

L1	1487	S ISCHEMIA AND GENE THERAPY
L2	317	S L1 AND BRAIN
L3	29	S L2 AND HIPPOCAMPUS
L4	26	DUP REMOVE L3 (3 DUPLICATES REMOVED)
L5	438	S SENDAI VIRUS AND VECTOR
L6	0	S L5 AND GAENE THERAPY
L7	191	S L5 AND GENE THERAPY
L8	133	DUP REMOVE L7 (58 DUPLICATES REMOVED)
L9	10	S L8 AND ISCHEMIA

(FILE 'HOME' ENTERED AT 14:27:27 ON 10 JAN 2003)

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE' ENTERED AT 14:27:45 ON 10 JAN 2003

L1	1487	S ISCHEMIA AND GENE THERAPY
L2	317	S L1 AND BRAIN
L3	29	S L2 AND HIPPOCAMPUS
L4	26	DUP REMOVE L3 (3 DUPLICATES REMOVED)

=> d ibib abs 1-10

L9 ANSWER 1 OF 10 MEDLINE
ACCESSION NUMBER: 2002276583 MEDLINE
DOCUMENT NUMBER: 22011794 PubMed ID: 12016262
TITLE: Angiogenic **gene therapy** for experimental critical limb **ischemia**: acceleration of limb loss by overexpression of vascular endothelial growth factor 165 but not of fibroblast growth factor-2.
AUTHOR: Masaki Ichiro; Yonemitsu Yoshikazu; Yamashita Akihisa; Sata Shihoko; Tanii Mitsugu; Komori Kimihiro; Nakagawa Kazunori; Hou Xiaogang; Nagai Yoshiyuki; Hasegawa Mamoru; Sugimachi Keizo; Sueishi Katsuo
CORPORATE SOURCE: Department of Pathology, Graduate School of Medical Sciences, Kyushu University, Fukuoka, Japan.
SOURCE: CIRCULATION RESEARCH, (2002 May 17) 90 (9) 966-73. Journal code: 0047103. ISSN: 1524-4571.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200205
ENTRY DATE: Entered STN: 20020518
Last Updated on STN: 20020529
Entered Medline: 20020528

AB Recent studies suggest the possible therapeutic effect of intramuscular vascular endothelial growth factor (VEGF) gene transfer in individuals with critical limb **ischemia**. Little information, however, is available regarding (1) the required expression level of VEGF for therapeutic effect, (2) the related expression of endogenous angiogenic factors, including fibroblast growth factor-2 (FGF-2), and (3) the related adverse effects due to overexpression of VEGF. To address these issues, we tested effects of overexpression of VEGF165 using recombinant **Sendai virus** (SeV), as directly compared with FGF-2 gene transfer. Intramuscular injection of SeV strongly boosted FGF-2, resulting in significant therapeutic effects for limb salvage with increased blood perfusion associated with enhanced endogenous VEGF expression in murine models of critical limb **ischemia**. In contrast, VEGF165 overexpression, 5-times higher than that of baseline on day 1, also strongly evoked endogenous VEGF in muscles, resulting in an accelerated limb amputation without recovery of blood perfusion. Interestingly, viable skeletal muscles of either VEGF165- or FGF-2-treated ischemic limbs showed similar platelet-endothelial cell adhesion molecule-1-positive vessel densities. Maturation of newly formed vessels suggested by smooth muscle cell actin-positive cell lining, however, was significantly disturbed in muscles with VEGF. Further, therapeutic effects of FGF-2 were completely diminished by anti-VEGF neutralizing antibody in vivo, thus indicating that endogenous VEGF does contribute to the effect of FGF-2. These results suggest that VEGF is necessary, but should be delicately regulated to lower expression to treat ischemic limb. The therapeutic effect of FGF-2, associated with the harmonized angiogenic effects seen with endogenous

VEGF, provides important insights into therapeutic angiogenesis.

L9 ANSWER 2 OF 10 MEDLINE
ACCESSION NUMBER: 2001645599 MEDLINE
DOCUMENT NUMBER: 21552996 PubMed ID: 11696476
TITLE: Therapeutic angiogenesis induced by human hepatocyte growth factor gene in rat diabetic hind limb **ischemia** model: molecular mechanisms of delayed angiogenesis in diabetes.
AUTHOR: Taniyama Y; Morishita R; Hiraoka K; Aoki M; Nakagami H; Yamasaki K; Matsumoto K; Nakamura T; Kaneda Y; Ogihara T
CORPORATE SOURCE: Department of Geriatric Medicine, Division of Gene Therapy Science, Biomedical Research Center, Osaka University Medical School, Suita, Japan.
SOURCE: CIRCULATION, (2001 Nov 6) 104 (19) 2344-50. Journal code: 0147763. ISSN: 1524-4539.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200112
ENTRY DATE: Entered STN: 20011108
Last Updated on STN: 20020123
Entered Medline: 20011204

AB BACKGROUND: Because no study has documented the angiogenic properties of hepatocyte growth factor (HGF) in a diabetes model, we examined the feasibility of **gene therapy** using HGF to treat peripheral arterial disease in diabetes. METHODS AND RESULTS: Because intramuscular injection of luciferase plasmid by the hemagglutinating virus of Japan (HVJ)-liposome method had much higher efficiency than injection of naked plasmid, we used the HVJ-liposome method to transfect the human HGF gene into the rat diabetic hindlimb model. As expected, transfection of human HGF **vector** resulted in a significant increase in blood flow as assessed by laser Doppler imaging and capillary density, even in the diabetes model, accompanied by the detection of human HGF protein. Interestingly, the degree of natural recovery of blood flow was significantly greater in nondiabetic rats than in diabetic rats. Thus, in an in vitro culture system, we further studied the molecular mechanisms of how diabetes delayed angiogenesis. Importantly, high-D-glucose treatment of endothelial cells resulted in a significant decrease in matrix metalloproteinase (MMP)-1 protein and ets-1 expression in human aortic endothelial cells. Similarly, high D-glucose significantly decreased mRNA and protein of HGF in endothelial cells. Downregulation of MMP-1 and ets-1 by high D-glucose might be due to a significant decrease in HGF, because HGF stimulated MMP-1 production and activated ets-1. CONCLUSIONS: Overall, intramuscular injection of human HGF plasmid induced therapeutic angiogenesis in a rat diabetic ischemic hindlimb model as a potential therapy for peripheral arterial disease. The delay of angiogenesis in diabetes might be due to downregulation of MMP-1 and ets-1 through a decrease in HGF by high D-glucose.

L9 ANSWER 3 OF 10 MEDLINE
ACCESSION NUMBER: 1999307583 MEDLINE
DOCUMENT NUMBER: 99307583 PubMed ID: 10377521
TITLE: **Gene therapy** using HVJ-liposomes: the best of both worlds?.
AUTHOR: Kaneda Y; Saeki Y; Morishita R
CORPORATE SOURCE: Division of Gene Therapy Science, Osaka University School of Medicine, 2-2 Yamada-oka, Suita, Osaka 565-0871, Japan..

kaneday@gts.med.osaka-u.ac.jp
SOURCE: MOLECULAR MEDICINE TODAY, (1999 J[illegible]) 5 (7) 298-303. Ref:
Journal code: 9508560. ISSN: 1357-4310.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199909
ENTRY DATE: Entered STN: 19991005
Last Updated on STN: 19991005
Entered Medline: 19990921

AB A new concept for the development of novel **vectors** is to overcome the limitations of individual **vectors** by combining them. The HVJ-liposome was developed by combining liposomes with fusion proteins derived from the hemagglutinating virus of Japan (HVJ), also known as **Sendai virus**. Gene transfer in vivo using this delivery system can be repeated because it is much less immunogenic and cytotoxic than other viral-**vector** systems. By coupling the Epstein-Barr virus (EBV) replicon apparatus with HVJ-liposomes, transgene expression can be sustained in vitro and in vivo. In animal models, this system has shown promise for several diseases, including cancer and cardiovascular disease.

L9 ANSWER 4 OF 10 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:408819 CAPLUS
DOCUMENT NUMBER: 136:396960
TITLE: Paramyxovirus **vector** encoding angiogenesis gene and use for tissue-specific gene transfer and **gene therapy**
INVENTOR(S): Yonemitsu, Yoshikazu; Sueishi, Katsuo; Fukumura, Masayuki; Hou, Xiaogang; Hasegawa, Mamoru
PATENT ASSIGNEE(S): Dnavec Research Inc., Japan
SOURCE: PCT Int. Appl., 94 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002042481	A1	20020530	WO 2001-JP10323	20011127
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2002024113	A5	20020603	AU 2002-24113	20011127

PRIORITY APPLN. INFO.: JP 2000-359374 A 20001127
WO 2001-JP10323 W 20011127

AB Paramyxovirus **vector** encoding an angiogenesis gene and use for tissue-specific angiogenesis gene transfer and **gene therapy** for **ischemia** are disclosed. **Sendai virus** (SeV) lacking the F gene is used. Recent studies suggest the possible therapeutic effect of i.m. vascular endothelial growth factor (VEGF) gene transfer in individuals with crit. limb **ischemia**. Little information, however, is available regarding (1) the required expression level of VEGF for therapeutic effect, (2) the related

expression of endogenous angiogenic factors, including fibroblast growth factor-2 (FGF-2), and (3) the related adverse effects due to overexpression of VEGF. To address these issues, the authors tested effects of overexpression of VEGF165 using recombinant **Sendai virus** (SeV), as directly compared with FGF-2 gene transfer. I.m. injection of SeV strongly boosted FGF-2, resulting in significant therapeutic effects for limb salvage with increased blood perfusion assocd. with enhanced endogenous VEGF expression in murine models of crit. limb **ischemia**. In contrast, VEGF165 overexpression, 5-times higher than that of baseline on day 1, also strongly evoked endogenous VEGF in muscles, resulting in an accelerated limb amputation without recovery of blood perfusion. Interestingly, viable skeletal muscles of either VEGF165- or FGF-2-treated ischemic limbs showed similar platelet-endothelial cell adhesion mol.-1-pos. vessel densities. Maturation of newly formed vessels suggested by smooth muscle cell actin-pos. cell lining, however, was significantly disturbed in muscles with VEGF. Further, therapeutic effects of FGF-2 were completely diminished by anti-VEGF neutralizing antibody in vivo, thus indicating that endogenous VEGF does contribute to the effect of FGF-2. These results suggest that VEGF is necessary, but should be delicately regulated to lower expression to treat ischemic limb. The therapeutic effect of FGF-2, assocd. with the harmonized angiogenic effects seen with endogenous VEGF, provides important insights into therapeutic angiogenesis.

REFERENCE COUNT: 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 5 OF 10 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2002:90572 CAPLUS
DOCUMENT NUMBER: 136:139817
TITLE: Negative-sense RNA virus **vector** for nerve cell targeting
INVENTOR(S): Fukumura, Masayuki; Asakawa, Makoto; Hasegawa, Mamoru; Shirakura, Masayuki
PATENT ASSIGNEE(S): Dnavec Research, Inc., USA
SOURCE: U.S. Pat. Appl. Publ., 25 pp., Cont.-in-part of U.S. Ser. No. 720,979.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002012995	A1	20020131	US 2001-843922	20010430
WO 2000001837	A1	20000113	WO 1999-JP3552	19990701

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: JP 1998-204333 A 19980703
WO 1999-JP3552 W 19990701
US 2001-720979 A2 20010307

AB Use of a neg.-sense RNA virus **vector** has enabled transfer of nucleic acid into nerve cells. The method of this invention can be used

for introducing a gene efficiently into nerve cells including central nervous system tissue in **gene therapy**, etc.

L9 ANSWER 6 OF 10 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:757674 CAPLUS
DOCUMENT NUMBER: 136:395641
TITLE: Heat shock protein 70 gene transfection protects mitochondrial and ventricular function against **ischemia**-reperfusion injury
AUTHOR(S): Jayakumar, Jay; Suzuki, Ken; Sammut, Ivan A.; Smolenski, Ryszard T.; Khan, Mak; Latif, Najma; Abunasra, Haitham; Murtuza, Bari; Amrani, Mohamed; Yacoub, Magdi H.
CORPORATE SOURCE: Department of Cardiothoracic Surgery, National Heart and Lung Institute, Imperial College School of Medicine, Royal Brompton and Harefield Hospital, Harefield, UB9 6JH, UK
SOURCE: Circulation (2001), 104(12, Suppl.), I303-I307
CODEN: CIRCAZ; ISSN: 0009-7322
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Upregulation of heat shock protein 70 (HSP70) is beneficial in cardioprotection against **ischemia**-reperfusion injury, but the mechanism of action is unclear. We studied the role of HSP70 overexpression through **gene therapy** on mitochondrial function and ventricular recovery in a protocol that mimics clin. donor heart preservation. Hemagglutinating virus of Japan (HVJ)-liposome technique was used to transfect isolated rat hearts via intracoronary infusion of either the HSP70 gene (HSP group, n = 16) or no gene (CON group, n = 16), which was heterotopically transplanted into recipient rats. Four days after surgery, hearts were either perfused on a Langendorff app. for 30 min at 37.degree.C (preischemia studies [n = 8/group]) or perfused for 30 min at 37.degree.C, cardioplegically arrested for 4 h at 4.degree.C, and reperfused for 30 min at 37.degree.C (postischemia studies [n = 8/group]). Western blotting and immunohistochem. confirmed HSP70 upregulation in the HSP group. Postischemic mitochondrial respiratory control indexes (RCIs) were significantly better preserved in HSP than in CON hearts: NAD+-linked RCI values were 9.54.+-.1.1 vs. 10.62.+-.0.46 before **ischemia** (NS) but 7.98.+-.0.69 vs. 1.28.+-.0.15 after **ischemia** (P<0.05), and FAD-linked RCI values were 6.87.+-.0.88 vs. 6.73.+-.0.93 before **ischemia** (NS) but 4.26.+-.0.41 vs. 1.34.+-.0.13 after **ischemia** (P<0.05). Postischemic recovery of mech. function was greater in HSP than in CON hearts: left ventricular developed pressure recovery was 72.4.+-.6.4% vs. 59.7.+-.5.3% (P<0.05), max. dP/dt recovery was 77.9.+-.6.6% vs. 52.3.+-.5.2% (P<0.05), and min. dP/dt recovery was 72.4.+-.7.2% vs. 54.8.+-.6.9% (P<0.05). Creatine kinase release in coronary effluent after reperfusion was 0.20.+-.0.04 vs. 0.34.+-.0.06 IU .cntdot. min-1 .cntdot. g wet wt-1 (P<0.05) in HSP vs. in CON hearts. HSP70 upregulation protects mitochondrial function after **ischemia** -reperfusion injury; this was assocd. with improved preservation of ventricular function. Protection of mitochondrial function may be important in the development of future cardioprotective strategies.
REFERENCE COUNT: 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 7 OF 10 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:580635 CAPLUS
DOCUMENT NUMBER: 135:353158
TITLE: **Gene therapy** for preventing neuronal death using hepatocyte growth factor: in vivo

TITLE: gene transfer of HGF to subarachnoid space prevents delayed neuronal death in gerbil hippocampal CA1 neurons
AUTHOR(S): Hayashi, K.; Morishita, R.; Nakagami, H.; Yoshimura, S.; Hara, A.; Matsumoto, K.; Nakamura, T.; Ogihara, T.; Kaneda, Y.; Sakai, N.
CORPORATE SOURCE: Department of Neurosurgery, Gifu University School of Medicine, Gifu, Japan
SOURCE: Gene Therapy (2001), 8(15), 1167-1173
CODEN: GETHEC; ISSN: 0969-7128
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English
AB To develop a novel strategy to prevent delayed neuronal death (DND) following transient occlusion of arteries, the gene of hepatocyte growth factor (HGF), a novel neurotropic factor, was transfected into the subarachnoid space of gerbils after transient forebrain **ischemia**. Importantly, transfection of HGF gene into the subarachnoid space prevented DND, accompanied by a significant increase in HGF in the cerebrospinal fluid. Prevention of DND by HGF is due to the inhibition of apoptosis through the blockade of bax translocation from the cytoplasm to the nucleus. HGF gene transfer into the subarachnoid space may provide a new therapeutic strategy for cerebrovascular disease.
REFERENCE COUNT: 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 8 OF 10 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2000:671906 CAPLUS
DOCUMENT NUMBER: 134:256670
TITLE: Developing a virosome-mediated gene delivery
AUTHOR(S): Kaneda, Yasufumi; Morishita, Ryuichi
CORPORATE SOURCE: Division of Gene Therapy Sciencey, Graduate School of Medicine, Osaka University, Suita, 565-0871, Japan
SOURCE: Proceedings of the International Symposium on Controlled Release of Bioactive Materials (2000), 27th, 171-172
CODEN: PCRMEY; ISSN: 1022-0178
PUBLISHER: Controlled Release Society, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A novel hybrid gene transfer **vector** was developed by combining viral and nonviral **vectors**. DNA-loaded liposomes consisting of phospholipids and cholesterol were prepd. by vortexing or reverse-phase evapn. The liposomes were fused with UV-inactivated HVJ (**Sendai virus**) to form the fusogenic viral-liposome, HVJ-liposome (400 to 500 nm in diam.). For more efficient gene delivery, lipid components of the liposomes were investigated and new anionic liposomes with a virus-mimicking lipid compn. (HVJ-AVE liposome) and HVJ-cationic liposomes were developed. For longterm gene expression, Epstein-Barr virus replicon **vector** was also developed. HVJ-liposome gene delivery system seem to be promising for the treatment of intractable human diseases.
REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 9 OF 10 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2000:34991 CAPLUS
DOCUMENT NUMBER: 132:74561
TITLE: Nerve cells-specific gene transfer using (-)-strand RNA virus **vector**
INVENTOR(S): Fukumura, Masayuki; Asakawa, Makoto; Hasegawa, Mamoru

PATENT ASSIGNEE(S): Dnavec Research Inc., Japan
SOURCE: PCT Int. Appl., 39 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000001837	A1	20000113	WO 1999-JP3552	19990701
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2336472	AA	20000113	CA 1999-2336472	19990701
AU 9943955	A1	20000124	AU 1999-43955	19990701
EP 1094115	A1	20010425	EP 1999-926878	19990701
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
US 2002012995	A1	20020131	US 2001-843922	20010430

PRIORITY APPLN. INFO.: JP 1998-204333 A 19980703
WO 1999-JP3552 W 19990701
US 2001-720979 A2 20010307

AB A recombinant **vector** derived from a (-)-strand RNA virus, such as **Sendai virus** (SeV) of Paramyxoviridae, is used for nerve cells-specific gene transfer for **gene therapy**. The method was demonstrated by introducing the gene for green fluorescent protein (GFP) into the cultured nerve cell lines, primary nerve cell culture, or cerebral ventricles of rat or mice. Expression of the gene for FGF-1 or FGF-5 from **vector** FGF-1-SeV or FGF-5-SeV inoculated into the cerebral ventricles of mice, and their effects on feed redn. were shown. Hippocampus ependymal cell.

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 10 OF 10 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1997:242449 CAPLUS
DOCUMENT NUMBER: 126:287832
TITLE: Efficient gene transfer method into the whole heart through the coronary artery with hemagglutinating virus of Japan liposome
AUTHOR(S): Sawa, Yoshiki; Kadoba, Keishi; Suzuki, Ken; Bai, Hong-Zhi; Kaneda, Yasufumi; Shirakura, Ryota; Matsuda, Hikaru
CORPORATE SOURCE: First Department of Surgery, Osaka University Medical School, Suita, 565, Japan
SOURCE: Journal of Thoracic and Cardiovascular Surgery (1997), 113(3), 512-519
CODEN: JTCSAQ; ISSN: 0022-5223
PUBLISHER: Mosby-Year Book
DOCUMENT TYPE: Journal
LANGUAGE: English

AB To confirm gene transfer techniques esp. into the whole heart, we tried out a gene transfer method involving liposome with the viral envelope hemagglutinating virus of Japan liposome as an alternative to existing techniques such as cationic lipofection or other viral **vectors**.

For this study, hemagglutinating virus of Japan liposome (H group) or cationic liposome(L group) was used to compare the efficacy of gene transfection of oligonucleotide labeled with fluorescein isothiocyanate and cDNA of .beta.-galactosidase and human manganese-superoxide dismutase.

Fluorescein-labeled oligonucleotide, cDNA of .beta.-galactosidase, or manganese-superoxide dismutase was complexed with liposomes, DNA-binding nuclear protein, and the viral protein coat of hemagglutinating virus of Japan. After donor rat hearts arrested by cardioplegia had been harvested, the coronary artery during cardioplegic arrest was infused via an aortic cannula with the liposome-gene complex. Next, the hearts were transplanted into the abdomen of recipient rats of the same strain, and all recipients were put to death after 3 days of transfection. Fluorescein isothiocyanate was detected in the nuclei of more than 70% of the myocytes (75% .+-. 14%) in the H group compared with fewer than 10% in the L group (7% .+-. 5%). The intensity of fluorescein isothiocyanate was significantly higher in the H group (979.+-.112 FI) than in the L group (116.+-.68 FI). .beta.-Galactosidase was expressed in the cytosol of more than 50% of the myocytes in the H group (61% .+-. 7%) compared with none in the L group (0%). After 3 days of gene transfection, and when exposed to **ischemia** (30 min, 37.degree.) and reperfusion (30 min, 37.degree.) with Langendorff app., the hearts transfected with manganese-superoxide dismutase (S group) showed a significantly higher percentage of recovery of left ventricular end-diastolic pressure (S vs. C, 86% .+-. 3% vs. 54% .+-. 12%) and coronary flow (98% .+-. 2% vs. 66% .+-. 12%) than did the control hearts (C group). Western blotting anal. showed an apparent increased expression of manganese-superoxide dismutase in the hearts transfected with manganese-superoxide dismutase compared with the control hearts. These results clearly demonstrated that the donor hearts were transfected with fluorescein-labeled oligonucleotide and the .beta.-galactosidase gene as a result of coronary infusion of the hemagglutinating virus of Japan liposome during cardioplegic arrest at the time of harvest. Furthermore, the hearts transfected with manganese-superoxide dismutase showed significant improvement in tolerance against **ischemia**-reperfusion injury. We believe that this method represents a novel in vivo gene transfer technique for the heart and thus may provide a new tool for research and therapy of heart transplantation.

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